case of epidemic mucormycosis; we stress the necessity to control all surfaces of a room in which a patient has been infected.

V. Levy
B. Rio
A. Bazarbachi
M. Hunault
A. Delmer
R. Zittoun

Service d'Hématologie, Hôtel-Dieu, Paris, France

V. Blanc

Laboratoire de Microbiologie, Hôtel-Dieu, Paris, France

M. Wolff

Service de Reanimation, Hôpital Bichat, Paris, France

References

1. Kwong-Chung KJ, Bennett JE: "Medical Mycology." Philadelphia: Lea & Febiger, 1992.
2. St-Germain G, Robert A, Ishak M, Tremblay C, Claveau S: Infection due to *Rhizomucor pusillus:* Report of four cases in patients with leukemia and review. Clin Infect Dis 16:640–645, 1992.
3. Pagano L, Ricci P, Nosari A, Tonso A, Buelli M, Montillo M, Cudillo L, Cenacchi A, Savignana C, Meillo L, Chierichini A, Marra R, Bucaneve G, Leone G, Del Gavero A: Fatal haemoptysis in pulmonary filamentous mycosis: An underevaluated cause of death in patients with acute leukaemia in haematological complete remission. A retrospective study and a review of the literature. Br J Haematol 89:500–505, 1995.
4. Ng TTC, Robson GD, Dennings DW: Hydrocortisone-enhanced growth of *Aspergillus* spp.: Implication for pathogenesis. Microbiology 140:2475–2479, 1994.
5. McWhinney PHM, Kibbler CC, Hamon MD: Progress in the diagnosis and management of aspergillosis in bone marrow transplantation: 13 years experience. Clin Infect Dis 17:397–404, 1993.

Translocation 21;22 May Be Involved in Control of Differentiation in Erythroleukemia

To the Editor: A 61-year-old man was admitted to Urafune Hospital of Yokohama City University with severe pancytopenia in September, 1994. The hemoglobin was 7.3 g/dl, white blood cell (WBC) count 1,000/μl with 87% lymphocytes, platelet count 25,000/μl, and erythroblast count 4/100 WBC. Bone marrow examination showed 52.4% erythroblasts, 37.2% myeloblasts of all nucleated cells, and 92.1% myeloblasts of nonerythroid cells. The blasts were negative to periodic acid-Schiff stain and α-naphtylbutylate esterase and positive to peroxidase. Morphology study showed that the blasts had large azurophilic granules, an atypical nucleus, and frequently Auer rods [Fig. 1]. Many erythroblasts showed megaloblastoid changes. Immunophenotypic analysis of the blasts revealed a positive reaction for CD13, CD33, and HLA-DR. Cytogenetic analysis was performed on bone marrow cells using G banding. All of 16 metaphases revealed a 21;22 translocation: 46,XY,t(21;22)(22q;q1?). On the basis of these results, we diagnosed acute non-lymphocytic leukemia (FAB-M6) and treated the patient with daunorbicin, cytosine arabinoside, 6-mercaptopurine, and prednisolone. The patient died of sepsis in October, 1994. We tried to examine bone marrow mononuclear cells (cryoreserved on admission) with regard to AML1 and major *bcr/abl* fusion genes by fluorescence in situ hybridization (FISH), but these genes could not be detected.

Olopade et al. [1] reported that erythroleukemia showed chromosomal abnormalities in over 70% of cases and generally clonal abnormalities led to loss of all or part of the long arm of chromosome 5 and/or chromosome 7. Chromosomal abnormalities on a 21;22 translocation have been previously reported in three cases of chronic myelogenous leukemia [2–4] but have not been noted in acute leukemia. One of the translocation in M2 is the t(8;21)(q22;q22), which has recently been shown to involve the AML1 gene at 21q22, and it has been suggested that the AML1 gene may be involved in control of cellular proliferation and/or differentiation [5]. Although we could not detect AML1 and major *bcr/abl* fusion genes by FISH, it was thought that in this case a 21;22 translocation caused differentiation in leukemic cells with azurophilic granules.

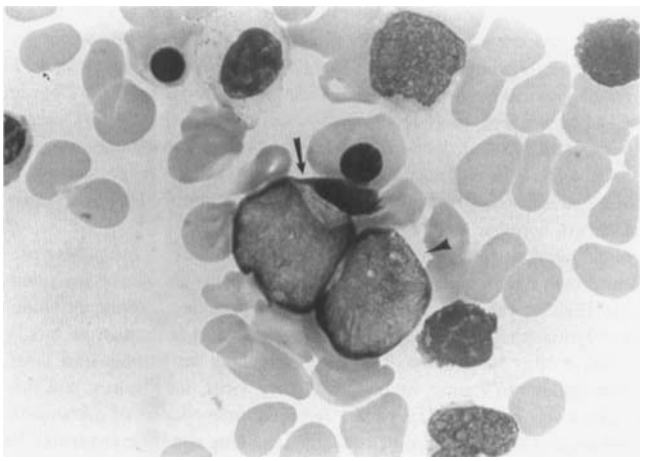

Fig. 1. Blast cells in bone marrow, with Auer rods (arrow) and azurophilic granules (arrowhead). (Wright-Giemsa stain, ×1,000.)

Shin Fujisawa
Shigeki Motomura
Katsumichi Fujimaki
Takao Okubo

First Department of Internal Medicine, Urafune Hospital, Yokohama City University School of Medicine, Yokohama, Japan

References

1. Olopade OI, Thangavelu M, Larson RA, Mick R, Kowal-Vern A, Schumacher HR, Le Beau MM, Vardiman JW, Rowley JD: Clinical, morphologic, and cytogeneic characteristics of 26 patients with acute erythroblastic leukemia. Blood 80:2873–2882, 1992.
2. Pinkerton PH, London B, Senn JS: Chronic myeloid leukemia with a philadelphia chromosome involving a t(21;22). Cancer Gene Cytogenet 12:175–178, 1984.
3. Bartram CR, Anger B, Carbonell F, Kleihauer E: Involvement of chromosome 9 in variant Ph^1 translocation. Leuk Res 9:1133–1137, 1985.
4. Bottura C, Coutinho V: G/C translocation and chronic myelocytic leukaemia. Blut 29:216–218, 1974.
5. Miyoshi H, Shimizu K, Kozu T, Maseki N, Kaneko Y, Ohki M: t(8;21) breakpoints on chromosome 21 in acute myeloid leukemia are clustered within a limited region of a single gene, AML1. Proc Natl Acad Sci USA 88:10431–10435, 1991.

Hodgkin's Lymphoma in a Cyclist Treated With Growth Hormone

To the Editor: The misuse of growth hormone (GH) to enhance athletic performance may become a significant challenge to the sports world. It is apparent that doping is increasingly used also by amateur and non-competi-

tive athletes; younger people are often involved. For this reason there is growing concern about long-term adverse effects of these substances.

We recently observed a case of Hodgkin's lymphoma occurring in a 31-year-old, non-smoking, white male patient. From the ages of 10 to 27 years he had been a semi-professional cyclist, riding competitively with a team. He reported habitual, long-lasting consumption of testosterone and other anabolic steroids, caffeine, and amphetamines. Moreover, he reported at least four subsequent administrations of high (supra-therapeutical) doses of growth hormone.

Four years after withdrawal from competitive activity, the patient presented with right inguinal lymphoadenopathy, in the absence of any symptom. Histological examination revealed lymphocyte-predominant Hodgkin's lymphoma. Total-body axial tomography and bone marrow biopsy excluded other localizations (IA stage). Serology for Epstein-Barr virus, cytomegalovirus, human immunodeficiency virus, herpesvirus, and toxoplasma was negative. The patient received three courses of adriamycin, bleomycin, vinblastine, and dacarbazine obtaining complete remission; he will also receive local radiotherapy.

Growth hormone is mitogenic for both the rat T-cell lymphoma line Nb2 and human lymphoid IM9 cells; it enhances the proliferation of human leukemic blasts [1] and increases the incidence of virus-induced B-cell lymphoma [2]. Recent reviews indicate that leukemia and related malignancies are frequently observed in GH-deficient children after GH therapy [3,4]. In our case, there was no evidence of additional risk factors for lymphoma, except for the doping. The latency period was comparable to those found in the literature for leukemia in GH-treated patients.

The use of doping in sports is probably more a moral issue than a medical one. However, the coexistence of vested interest for athletes and team managers in the use of non-legal pharmaceuticals may enlarge the field of action of criminality in sports. The suspected relationship between GH use and hematological malignancies represents a further, strong reason to discourage this practice.

Nicola Magnavita

Institute of Occupational Medicine,

Luciana Teofili
Giuseppe Leone

Department of Hematology, Catholic University School of Medicine, Rome, Italy

References

1. Estrov Z, Meir R, Barak Y, Zaizov R, Zadik Z: Human growth hormone and insulin-like growth factor-1 enhance the proliferation of human leukemic blasts. J Clin Oncol 9:394–399, 1991.
2. Murphy WJ, Durum SK, Anver M, Frazier M, Longo DL: Recombinant human growth hormone promotes human lymphocyte engraftment in immunodeficient mice and results in an increased incidence of human Epstein Barr virus-induced B-cell lymphoma. Brain Behav Immun 6:355–364, 1992.
3. Watanabe S, Mizuno S, Oshima LH, Tsunematsu Y, Fujimoto J, Komiyama A: Leukemia and other malignancies among GH users. J Pediatr Endocrinol 6:99–108, 1993.
4. Magnavita N, Sica S, Di Mario A, Leone G: Concurrent epiphyseal fracture and leukemia in a patient treated with growth hormone. Am J Hematol 51:96, 1996.

Physiological Neutrophilia Is Associated With Elevated Serum Level of Macrophage Colony-Stimulating Factor (M-CSF)

To the Editor: Cincotta et al. [1] reported that physiological neutrophilia of pregnancy is not associated with a rise in plasma granulocyte colony-stimulating factor (G-CSF). We agree with this observation. Recently our colleagues reported that serum levels of macrophage colony-stimulating factor (M-CSF) are correlated with the presence of neutrophilia during pregnancy [2,3]. M-CSF plays an important role in trophoblast development and hormonal regulation of pregnancy. In early and late pregnancy, serum M-CSF levels are elevated along with the white blood cell count [4], and soon after delivery M-CSF levels return to normal.

The report of Cincotta et al. [1] about normal G-CSF levels during pregnancy is quite important. When we encounter pregnant patients who may have infections, white blood cell count is not necessarily a useful parameter for diagnosis. C-reactive protein or other inflammatory parameters should be helpful, but serum levels of G-CSF and M-CSF should also be taken into consideration to determine the clinical situation of the pregnant woman.

Kiyohiko Hatake
Yasusada Miura

Division of Hematology, Department of Medicine, Jichi Medical School, Tochigi

Kazuo Motoyoshi

Third Department of Internal Medicine, National Defence Medical College, Saitama, Japan

References

1. Cincotta R, Balloch A, Metz J, Layton JE, Lieschke GJ: Physiological neutrophilia of pregnancy is not associated with a rise in plasma granulocyte colony-stimulating factor (G-CSF) Am J Hematol 48:288–289, 1995.
2. Hanamura T, Motoyoshi K, Yoshida K, Saito M, Miura Y, Kawashima T, Nishida M, Takaku F: Quantitation and identification of human monocytic colony-stimulating factor in human serum by enzyme-linked immunosorbent assay. Blood 72:886–890, 1988.
3. Suzu S, Yanai N, Sato-Somoto J, Yamada M, Kawashima T, Hanamura T, Nagata N, Takaku F, Motoyoshi K: Characterization of macrophage colony-stimulating factor in body fluids by immunoblot analysis. Blood 77:2160–2164, 1991.
4. Praloran V, Coupey L, Donnard M, Berrada L, Naud MF: Elevation of serum M-CSF concentrations during pregnancy and ovarian hyperstimulation. Br J Haematol 86:675–677, 1994.

Kung Fu Phlebitis: An Unusual Presentation of Mondor's Disease

To the Editor: Mondor's disease, or superficial thrombophlebitis of the chest wall, is a relatively uncommon condition that has been described primarily in young and middle-aged women [1]. We report an unusual presentation of Mondor's disease in a man.

A 37-year-old man who was generally in good health presented to our outpatient clinic with a complaint of sharp, intermittent, right anterior chest wall pain that had begun 4 weeks previously. The pain was precipitated by abduction and extension of the right shoulder, and improved within minutes of repositioning the arm.

Physical examination revealed palpable cords originating near the right nipple and radiating inferiorly along the course of the thoracoepigastric vein and laterally along the course of the lateral thoracic vein. There was mild tenderness to palpation but no erythema or edema. Laboratory testing showed normal values for prothrombin time, activated partial thromboplastin time, fibrinogen, thrombin time, plasminogen, antithrombin III, protein C, protein S, dilute Russell viper venom time, and anticardiolipin antibodies. A polymerase chain reaction for factor V Leiden [2] was negative. A